WHAT IS CLAIMED IS:

1. A method of inhibiting a cancer cell comprising administering to the cancer cell a composition comprising a PPT1 modulator in an amount effective to reduce PPT1 activity level.

2. The method of claim 1, wherein inhibiting a cancer cell comprises altering proliferation, metastasis, contact inhibition, soft agar growth, cell cycle regulation, tumor formation, tumor progression, differentiation, programmed cell death, or tumor invasion.

3. The method of claim 1, wherein the PPT1 modulator comprises a proteinaceuous composition.

4. The method of claim 3, wherein the modulator competitively binds to PPT1.

5. The method of claim 4, wherein the modulator is an antagonist of PPT1.

6. The method of claim 1, wherein the modulator decreases the amount of PPT1.

7. The method of claim 1, wherein the modulator inhibits expression of PPT1.

8. The method of claim 4, wherein the modulator is at least one peptide or peptide mimetic that selectively interacts with PPT1.

9. The method of claim 8, wherein the modulator is at least one peptide that selectively interacts with PPT1.

10. The method of claim 8, wherein the modulator is at least one peptide mimetic that selectively interacts with PPT1.

11. The method of claim 9, wherein the peptide comprises at least or at most 5 contiguous amino acids from SEQ ID NO:3.

12. The method of claim 11, wherein the peptide comprises the sequence VKIKK.

13. The method of claim 9, wherein the peptide comprises at least or at most 5 contiguous amino acids from SEQ ID NO:4.

14. The method of claim 13, wherein the peptide comprises the sequence YCWLR.

15. The method of claim 8, wherein the peptide or peptide mimetic is attached to a lipid component.

16. The method of claim 15, wherein the lipid component is a fatty acid.

17. The method of claim 16, wherein the fatty acid is unbranched.

18. The method of claim 15, wherein the lipid component is 8 to 30 carbons long.

19. The method of claim 18, wherein there is a double bond between C4 and C5.

20. The method of claim 15, wherein the peptide or peptide mimetic is attached to the lipid component through a non-hydrolyzable link.

21. The method of claim 15, wherein the lipid component comprises an oxime ether.

22. The method of claim 12, wherein the peptide is DAP1.

23. The method of claim 22, wherein DAP1 is in an α-ketoamide form.

24. The method of claim 8, wherein the modulator of PPT1 is a peptide mimetic.

25. The method of claim 24, wherein the modulator of PPT1 is a peptide mimetic of the amino acid sequence VKIKK.

26. The method of claim 24, wherein the modulator of PPT1 is a peptide mimetic of the amino acid sequence YCWLR.

27. The method of claim 24, wherein the peptide mimetic comprises a lipid component.

28. The method of claim 1, wherein the modulator of PPT1 is a nucleic acid containing a promoter operably linked to a PPT1 gene segment.

29. The method of claim 28, wherein the PPT1 gene segment is positioned, in reverse orientation, under the control of a promoter that directs expression of an antisense product.

30. The method of claim 28, wherein the nucleic acid encodes a ribozyme specific for an RNA transcript of PPT1 in a cell expressing an RNA transcript of PPT1.

31. The method of claim 4, wherein the modulator is an antibody composition comprising an antibody that recognizes PPT1.

32. The method of claim 1, further comprising administering to the cancer cell a composition comprising a chemotherapeutic drug.

33. The method of claim 1, wherein the cell is in a mammal.

34. A method of treating a subject with cancer comprising administering to the subject a PPT1 modulator in an amount effective to inhibit a cancer cell in the subject, thereby conferring a therapeutic benefit on the subject.

35. The method of claim 34, wherein the modulator is a peptide or peptide mimetic that selectively interacts with PPT1.

36. The method of claim 35, wherein the modulator is a peptide.

37. The method of claim 35, wherein the modulator is a peptide mimetic.

38. The method of claim 36, wherein the peptide comprises at least or at most 5 contiguous amino acids from SEQ ID NO:3.

39. The method of claim 38, wherein the peptide comprises the sequence VKIKK.

40. The method of claim 36, wherein the peptide comprises at least or at most 5 contiguous amino acids from SEQ ID NO:4.

41. The method of claim 40, wherein the peptide comprises the sequence YCWLR.

42. The method of claim 35, wherein the peptide or peptide mimetic is attached to a lipid component.

43. The method of claim 42, wherein the lipid component is a fatty acid.

44. The method of claim 39, wherein the peptide is DAP1.

45. The method of claim 44, wherein DAP1 is in an α-ketoamide form.

46. The method of claim 34, further comprising treating the subject with a chemotherapeutic drug.

47. A method of screening a candidate substance for anti-cancer activity comprising:

(i) contacting a cancer cell with the candidate substance; and

(ii) assaying the compound's ability to modulate PPT1.

48. The method of claim 47, wherein modulation of PPT1 comprises altering PPT1 expression, activity, or location.

49. The method of claim 47, wherein assaying the compound's ability to modulate PPT1 comprises assaying for apoptosis.

50. The method of claim 49, further comprising administering a chemotherapeutic agent to the cell.

51. The method of claim 50, wherein the chemotherapeutic agent is administered to the cell prior to assaying for apoptosis.

52. The method of claim 47, wherein the cell is contacted *in vitro*.

53. The method of claim 47, wherein the cell is contacted *in vivo*.

54. A pharmaceutical composition comprising a recombinant vector containing an PPT1 gene segment positioned in reverse orientation, under the control of a promoter that directs expression of an antisense product.

55. A pharmaceutical composition comprising a peptide or peptide mimetic that selectively binds to PPT1 and that is covalently attached to a lipid component through a non-hydrolyzable linkage.

56. A pharmaceutical composition comprising a peptide mimetic that selectively binds to PPT1 and that is covalently attached to a lipid component through a non-hydrolyzable linkage.

57. A method of screening for cancer or pre-cancer in a subject comprising:

a) obtaining a sample from the subject;

b) assaying the sample for PPT1 amount or activity level;

c) comparing the PPT1 amount or activity level of the subject to the PPT1 amount or activity level of a noncancerous sample, wherein elevated PPT1 amount or activity level may indicate cancer or pre-cancer in the subject.